



MEETING ABSTRACT

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Nicotinic augmentation of anti-inflammatory GSK3 β signaling

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Background

Glycogen synthesis kinase 3 β (GSK3 β) has been shown to be a critical mediator of the intensity and direction of the innate immune system responding to bacterial stimuli. Stimulation of the anti-cholinergic anti-inflammatory system by tobacco alkaloids (nicotine; cotinine) leads to phosphorylation and inactivation of GSK3 β and, subsequently, to immune suppression. This presentation will review the tobacco-induced dysregulation of GSK3 β signaling and provide insight into the increased susceptibility of smokers to multiple bacterial diseases, including those caused by *Mycobacterium tuberculosis*, *Legionella pneumophila*, and *Neisseria meningitidis*. The extensive ongoing efforts to exploit GSK3 β for its therapeutic potential in the control of infectious diseases will also be reviewed.

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